Refine Search

Search Results -

Terms	Documents
L7 and L6	39

Database: US Pre-Grant Publication Full-Text Database
US Patents Full-Text Database
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JPO Abstracts Database
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IBM Technical Disclosure Bulletins

Search: L8

Refine Search

Recall Text | Clear | Interrupt

Search History

DATE: Monday, April 03, 2006 Printable Copy Create Case

Set Name side by side	Query	Hit Count	Set Name result set
DB=PGPB; PLUR=YES; OP=OR			
L8	L7 and l6	39	L8
L7	uckun.in.	73	L7
L6	L5 and JaK-3 kinase	37403	L6
L5	c-jun inhibition	67314	L5
L4	L1 and (c-jun activation associated with DNA damage)	1	L4
L3	L2 and (DNA damage)	1	L3
L2	20030144178	1	L2
L1	20030144178	1	L1

END OF SEARCH HISTORY

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Search Results - Record(s) 1 through 10 of 39 returned.

☐ 1. Document ID: US 20060046972 A1

L8: Entry 1 of 39 File: PGPB Mar 2, 2006

PGPUB-DOCUMENT-NUMBER: 20060046972
PGPUB-FILING-TYPE:
DOCUMENT-IDENTIFIER: US 20060046972 A1

TITLE: Cytotoxic nucleoside analog compound 003 for treating cancer

PUBLICATION-DATE: March 2, 2006

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY
Uckun; Fatih M.	White Bear Lake	MN	US
Venkatachalam; Taracad	Maplewood	MN	US

US-CL-CURRENT: 514/51

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc | Ima

☐ 2. Document ID: US 20050277620 A1

L8: Entry 2 of 39 File: PGPB Dec 15, 2005

PGPUB-DOCUMENT-NUMBER: 20050277620
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20050277620 A1

TITLE: Aryl phosphate derivatives of d4T

PUBLICATION-DATE: December 15, 2005

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY
Uckun, Fatih M.	White Bear Lake	MN	US

US-CL-CURRENT: 514/86; 544/243

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc | Ima

☐ 3. Document ID: US 20050198696 A1

L8: Entry 3 of 39 File: PGPB Sep 8, 2005

PGPUB-DOCUMENT-NUMBER: 20050198696
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20050198696 A1

TITLE: Transgenic zebra fish embryo model for hematopoiesis and lymphoproliferative disorders

PUBLICATION-DATE: September 8, 2005

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY
Uckun, Fatih M.	White Bear Lake	MN	US
Benyumov, Alexey O.	Plymouth	MN	US

US-CL-CURRENT: 800/9; 800/20

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc | Image

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☐ 4. Document ID: US 20050196851 A1

L8: Entry 4 of 39 File: PGPB Sep 8, 2005

PGPUB-DOCUMENT-NUMBER: 20050196851
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20050196851 A1

TITLE: Crystal structure of the BTK kinase domain

PUBLICATION-DATE: September 8, 2005

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY
Uckun, Fatih M.	White Bear Lake	MN	US

US-CL-CURRENT: 435/194; 702/19

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc | Image

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☐ 5. Document ID: US 20050187233 A1

L8: Entry 5 of 39 File: PGPB Aug 25, 2005

PGPUB-DOCUMENT-NUMBER: 20050187233
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20050187233 A1

TITLE: JAK-3 inhibitors for treating allergic disorders

PUBLICATION-DATE: August 25, 2005

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY
Uckun, Faith M.	White Bear Lake	MN	US
Malaviya, Ravi	Shoreview	MN	US
Sudbeck, Elise A.	St. Paul	MN	US

US-CL-CURRENT: 514/266.3; 514/266.4

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc | Image

☐ 6. Document ID: US 20050143339 A1

L8: Entry 6 of 39 File: PGPB Jun 30, 2005

PGPUB-DOCUMENT-NUMBER: 20050143339
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20050143339 A1

TITLE: Aryl phosphate derivatives of D4T with potent anti-viral activity

PUBLICATION-DATE: June 30, 2005

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY
Uckun, Fatih	White Bear Lake	MN	US
Chen, Chun-Lin	Roseville	MN	US
Venkatachalam, Taracad K.	Maplewood	MN	US
Zhu, Zhoa-Hai	Shoreview	MN	US

US-CL-CURRENT: 514/50; 514/86

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc | Ima

☐ 7. Document ID: US 20050119322 A1

L8: Entry 7 of 39 File: PGPB Jun 2, 2005

PGPUB-DOCUMENT-NUMBER: 20050119322
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20050119322 A1

TITLE: Phorboxazole derivatives for treating cancer

PUBLICATION-DATE: June 2, 2005

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY
Uckun, Fatih M.	White Bear Lake	MN	US
Narla, Rama K.	Shoreview	MN	US
Forsyth, Craig	Roseville	MN	US
Lee, Chi Sing	Pokfulam Gardens	NY	CN
Ahmed, Feryan	Albany	IL	US
Cink, Russell Drew	Grayslake		US

US-CL-CURRENT: 514/375

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc | Ima

☐ 8. Document ID: US 20050075353 A1

L8: Entry 8 of 39 File: PGPB Apr 7, 2005

PGPUB-DOCUMENT-NUMBER: 20050075353
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20050075353 A1

TITLE: Quinazolines and therapeutic use thereof

PUBLICATION-DATE: April 7, 2005

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY
Uckun, Fatih M.	White Bear Lake	MN	US
Liu, Xing-Ping	Minneapolis	MN	US
Narla, Rama Krishna	St. Paul	MN	US

US-CL-CURRENT: 514/266.4; 544/293

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc | Ima

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☐ 9. Document ID: US 20040235815 A1

L8: Entry 9 of 39 File: PGPB Nov 25, 2004

PGPUB-DOCUMENT-NUMBER: 20040235815
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20040235815 A1

TITLE: Vanadium compounds for treating cancer

PUBLICATION-DATE: November 25, 2004

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY
Uckun, Faith M.	White Bear Lake	MN	US
Dong, Yanhong	Moundsview	MN	US
Gosh, Phalguni	Shoreview	MN	US

US-CL-CURRENT: 514/184; 546/2

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc | Ima

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☐ 10. Document ID: US 20040192711 A1

L8: Entry 10 of 39 File: PGPB Sep 30, 2004

PGPUB-DOCUMENT-NUMBER: 20040192711
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20040192711 A1

TITLE: Therapeutic compounds

PUBLICATION-DATE: September 30, 2004

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY
Uckun, Fatih M.	White Bear Lake	MN	US
Sudbeck, Elise A.	St. Paul	MN	US
Cetkovic, Marina	Maplewood	MN	US
Malaviya, Ravi	Shoreview	MN	US
Liu, Xing-Ping	Minneapolis	MN	US

US-CL-CURRENT: 514/266.1; 514/266.3, 514/266.4, 544/283, 544/287, 544/293

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw. Desc | Ima

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Terms	Documents
L7 and L6	39

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NEWS 4	DEC 23	New IPC8 SEARCH, DISPLAY, and SELECT fields in USPATFULL/ USPAT2
NEWS 5	JAN 13	IPC 8 searching in IFIPAT, IFIUDB, and IFICDB
NEWS 6	JAN 13	New IPC 8 SEARCH, DISPLAY, and SELECT enhancements added to INPADOC
NEWS 7	JAN 17	Pre-1988 INPI data added to MARPAT
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NEWS 11	FEB 21	STN AnaVist, Version 1.1, lets you share your STN AnaVist visualization results
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NEWS 13	FEB 22	The IPC thesaurus added to additional patent databases on STN
NEWS 14	FEB 22	Updates in EPFULL; IPC 8 enhancements added
NEWS 15	FEB 27	New STN AnaVist pricing effective March 1, 2006
NEWS 16	FEB 28	MEDLINE/LMEDLINE reload improves functionality
NEWS 17	FEB 28	TOXCENTER reloaded with enhancements
NEWS 18	FEB 28	REGISTRY/ZREGISTRY enhanced with more experimental spectral property data
NEWS 19	MAR 01	INSPEC reloaded and enhanced
NEWS 20	MAR 03	Updates in PATDPA; addition of IPC 8 data without attributes
NEWS 21	MAR 08	X.25 communication option no longer available after June 2006
NEWS 22	MAR 22	EMBASE is now updated on a daily basis
NEWS 23	APR 03	New IPC 8 fields and IPC thesaurus added to PATDPAFULL
NEWS 24	APR 03	Bibliographic data updates resume; new IPC 8 fields and IPC thesaurus added in PCTFULL

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=> file medline, biosis
COST IN U.S. DOLLARS

	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.63	0.63

FILE 'MEDLINE' ENTERED AT 17:03:30 ON 03 APR 2006

FILE 'BIOSIS' ENTERED AT 17:03:30 ON 03 APR 2006

=> s (c-jun activation) and inhibition
L1 67 (C-JUN ACTIVATION) AND INHIBITION

=> s JAK-3 inhibition
L2 4 JAK-3 INHIBITION

=> s l4 and l1
L4 NOT FOUND
The L-number entered could not be found. To see the definition of L-numbers, enter DISPLAY HISTORY at an arrow prompt (=>).

=> s l2 and l1
L3 0 L2 AND L1

=> d l2 ti abs ibib tot

L2 ANSWER 1 OF 4 MEDLINE on STN
TI Combined use of the JAK3 inhibitor CP-690,550 with mycophenolate mofetil to prevent kidney allograft rejection in nonhuman primates.
AB BACKGROUND: Immunosuppression via Janus kinase (**JAK**) 3 **inhibition** affords significant prolongation of allograft survival. We investigated the effects of an immunosuppressive regimen combining the JAK3 inhibitor CP-690,550 with mycophenolate mofetil (MMF) in nonhuman primates (NHPs). METHODS: Life-supporting kidney transplantations were performed between ABO-compatible, MLR-mismatched NHPs. Animals were treated orally twice a day with CP-690,550 and MMF (n=8) or MMF alone (n=2) and were euthanized at day 90 or earlier due to allograft rejection. RESULTS: Mean survival time (+/-SEM) in animals treated with MMF alone (23+/-1 days) was significantly extended in animals that concurrently received CP-690,550 (59.5+/-9.8 days, P=0.02). Combination animals exposed to higher levels of CP-690,550 had a significantly better survival (75.2+/-8.7 days) than animals that received less CP-690,550 (33.3+/-12.6 days, P=0.02). Three combination therapy animals were euthanized at day 90 with a subnormal renal function and early-stage acute graft rejection. Rejection, delayed by treatment, ultimately developed in other animals. Anemia and gastrointestinal intolerance was seen in combination therapy animals that otherwise did not show evidence of viral or bacterial infection besides signs consistent with subclinical pyelonephritis (n=3). One incidental lymphosarcoma was noted. CONCLUSIONS: Addition of CP-690,550 to MMF significantly improved allograft survival. The observed side effects appear amenable to improvements upon alteration of dosing strategies. Efficacy of this combination regimen suggests that it could become the backbone of calcineurin inhibitor-free regimens.
ACCESSION NUMBER: 2006014933 MEDLINE
DOCUMENT NUMBER: PubMed ID: 16378072
TITLE: Combined use of the JAK3 inhibitor CP-690,550 with

mycophenolate mofetil to prevent kidney allograft rejection in nonhuman primates.

AUTHOR: Borie Dominic C; Larson Michael J; Flores Mona G; Campbell Andrew; Rousvoal Geraldine; Zhang Sally; Higgins John P; Ball Douglas J; Kudlacz Elizabeth M; Brissette William H; Elliott Eileen A; Reitz Bruce A; Changelian Paul S

CORPORATE SOURCE: Transplantation Immunology Laboratory, Department of Cardiothoracic Surgery, Falk Cardiovascular Research Center, Stanford University School of Medicine, Stanford, CA 94305-5407, USA.. dborie@stanford.edu

SOURCE: Transplantation, (2005 Dec 27) Vol. 80, No. 12, pp. 1756-64.
Journal code: 0132144. ISSN: 0041-1337.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200601
ENTRY DATE: Entered STN: 20060111
Last Updated on STN: 20060201
Entered Medline: 20060131

L2 ANSWER 2 OF 4 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

TI Combined use of the JAK3 inhibitor CP-690,550 with mycophenolate mofetil to prevent kidney allograft rejection in nonhuman primates.

AB Background. Immunosuppression via Janus kinase (**JAK**) 3 **inhibition** affords significant prolongation of allograft survival. We investigated the effects of an immunosuppressive regimen combining the JAK3 inhibitor CP-690,550 with mycophenolate mofetil (MMF) in nonhuman primates (NHPs).Methods. Life-supporting kidney transplantations were performed between ABO-compatible, MLR-mismatched NHPs. Animals were treated orally twice a day with CP-690,550 and MMF (n=8) or MMF alone (n=2) and were euthanized at day 90 or earlier due to allograft rejection.Results. Mean survival time (+/- SEM) in animals treated with MMF alone (23 +/- 1 days) was significantly extended in animals that concurrently received CP-690,550 (59.5 +/- 9.8 days, P=0.02). Combination animals exposed to higher levels of CP-690,550 had a significantly better survival (75.2 +/- 8.7 days) than animals that received less CP-690,550 (33.3 +/- 12.6 days, P=0.02). Three combination therapy animals were euthanized at day 90 with a subnormal renal function and early-stage acute graft rejection. Rejection, delayed by treatment, ultimately developed in other animals. Anemia and gastrointestinal intolerance was seen in combination therapy animals that otherwise did not show evidence of viral or bacterial infection besides signs consistent with subclinical pyelonephritis (n=3). One incidental lymphosarcoma was noted.Conclusions. Addition of CP-690,550 to MMF significantly improved allograft survival. The observed side effects appear amenable to improvements upon alteration of dosing strategies. Efficacy of this combination regimen suggests that it could become the backbone of calcineurin inhibitor-free regimens.

ACCESSION NUMBER: 2006:197109 BIOSIS
DOCUMENT NUMBER: PREV200600206157

TITLE: Combined use of the JAK3 inhibitor CP-690,550 with mycophenolate mofetil to prevent kidney allograft rejection in nonhuman primates.

AUTHOR(S): Borie, Dominic C. [Reprint Author]; Larson, Michael J.; Flores, Mona G.; Campbell, Andrew; Rousvoal, Geraldine; Zhang, Sally; Higgins, John P.; Ball, Douglas J.; Kudlacz, Elizabeth M.; Brissette, William H.; Elliott, Eileen A.; Reitz, Bruce A.; Changelian, Paul S.

CORPORATE SOURCE: Stanford Univ, Med Ctr, Sch Med,Falk Cardiovasc Res Ctr, Dept Cardiothorac Surg,Transplantat Immunol Lab, 300 Pasteur Dr,Falk CVRB, Stanford, CA 94305 USA
dborie@stanford.edu

SOURCE: Transplantation (Hagerstown), (DEC 27 2005) Vol. 80, No. 12, pp. 1756-1764.
CODEN: TRPLAU. ISSN: 0041-1337.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 22 Mar 2006
Last Updated on STN: 22 Mar 2006

L2 ANSWER 3 OF 4 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI **JAK-3 inhibition** in human T cells abrogates IL-2 production and early T cell clustering: Evidence for an impaired early TCR-signalling.
ACCESSION NUMBER: 2001:396329 BIOSIS
DOCUMENT NUMBER: PREV200100396329
TITLE: **JAK-3 inhibition** in human T cells abrogates IL-2 production and early T cell clustering: Evidence for an impaired early TCR-signalling.
AUTHOR(S): Saeemann, M. D. [Reprint author]; Boehmig, G. A.; Krieger, P.-M. [Reprint author]; Diakos, C. [Reprint author]; Prieschl-Strassmeier, E.; Baumruker, T.; Hoerl, W. H.; Zlabinger, G. [Reprint author]
CORPORATE SOURCE: Institute of Immunology, University of Vienna, Vienna, Austria
SOURCE: Nephrology Dialysis Transplantation, (June, 2001) Vol. 16, No. 6, pp. A212. print.
Meeting Info.: Annual Congress of the European Renal Association and the European Dialysis and Transplant Association. Vienna, Austria. June 24-27, 2001. European Renal Association; European Dialysis and Transplant Association.
ISSN: 0931-0509.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 22 Aug 2001
Last Updated on STN: 22 Feb 2002

L2 ANSWER 4 OF 4 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
TI Prevention of fatal thromboembolism in mice by selectively targeting Jak 3 kinase in platelets with 4-(4'-Hydroxylphenyl)-amino-6,7-dimethoxyquinazoline (WHI-P131).
AB The quinazoline derivative, 4-(4'-Hydroxylphenyl)-amino-6,7-dimethoxyquinazoline (WHI-P131) is a rationally designed specific inhibitor of Janus Kinase 3. We sought to determine the effects of WHI-P131 on platelet activation and aggregation in vitro as well as bleeding time and thromboplastin-induced fatal thromboembolism in vivo. At low micromolar concentrations, WHI-P131 inhibited thrombin-induced signaling events, including degranulation/serotonin release, membrane ruffling, pseudopod formation, and translocation of cytoplasmic proteins to the Tx-soluble and insoluble cytoskeleton. Thrombin-induced tyrosine phosphorylation as well as membrane localization of Stat 1 and Stat3beta were also markedly inhibited by WHI-P131. WHI-P131 inhibited thrombin-induced (but not collagen-induced) platelet aggregation with an IC50 value of 1.5 muM. Jak 3 deficient mice also exhibited a decrease in thrombin-induced platelet aggregation, overall tyrosine phosphorylation and phosphorylation of Stat 1 and Stat3beta. WHI-P131 was not toxic to mice when administered systemically at dose levels ranging from 1 mg/kg to 250 mg/kg. Highly effective platelet inhibitory plasma concentrations (gtoreq10 muM) of WHI-P131 could be achieved in mice without toxicity. At nontoxic dose levels, WHI-P131 prolonged the tail bleeding time of mice in dose-dependent manner and improved survival in a mouse model of thromboplastin-induced generalized and fatal thromboembolism. The probability of EFS after the thromboplastin challenge was 10+-7% (median

survival time=2.5 min) for the vehicle-treated control group (N=20), 30+-15 (median survival time=5.3 min) for warfarin-treated control group (N=20) (P=0.001), and 30+-17% (median survival time =5.2 min) for the WHI-P131-treated test group (25 mg/kg dose level; N=10) (P=0.001) This present study significantly expands our knowledge of the importance of Jak3 and the Stat family proteins in platelets. To our knowledge, WHI-P131 is the first anti-thrombotic agent which prevents platelet aggregation by inhibiting Jak 3.

ACCESSION NUMBER: 2001:311605 BIOSIS
DOCUMENT NUMBER: PREV200100311605
TITLE: Prevention of fatal thromboembolism in mice by selectively targeting Jak 3 kinase in platelets with 4-(4'-Hydroxylphenyl)-amino-6,7-dimethoxyquinazoline (WHI-P131).
AUTHOR(S): Tibbles, Heather E. [Reprint author]; Vassilev, Alexei O. [Reprint author]; Wendorf, Heather [Reprint author]; Lorenz, David [Reprint author]; Zhu, Dan [Reprint author]; Waurzyniak, Barbara [Reprint author]; Liu, Xing-Ping [Reprint author]; Uckun, Fatih M. [Reprint author]
CORPORATE SOURCE: Parker Hughes Institute, St. Paul, MN, USA
SOURCE: Blood, (November 16, 2000) Vol. 96, No. 11 Part 1, pp. 273a. print.
Meeting Info.: 42nd Annual Meeting of the American Society of Hematology. San Francisco, California, USA. December 01-05, 2000. American Society of Hematology.
CODEN: BLOOAW. ISSN: 0006-4971.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 27 Jun 2001
Last Updated on STN: 19 Feb 2002

=> d l1 ti abs ibib 1-10

L1 ANSWER 1 OF 67 MEDLINE on STN
TI Tumorigenesis suppressor pdcd4 down-regulates mitogen-activated protein kinase kinase kinase kinase 1 expression to suppress colon carcinoma cell invasion.
AB Programmed cell death 4 (Pdcd4) suppresses neoplastic transformation by inhibiting the activation of c-Jun and consequently AP-1-dependent transcription. We report that Pdcd4 blocks **c-Jun activation** by inhibiting the expression of mitogen-activated protein kinase kinase kinase kinase 1 (MAP4K1)/hematopoietic progenitor kinase 1, a kinase upstream of Jun N-terminal kinase (JNK). cDNA microarray analysis of Pdcd4-overexpressing RKO human colon carcinoma cells revealed MAP4K1 as the sole target of Pdcd4 on the JNK activation pathway. Cotransfection of a MAP4K1 promoter-reporter with Pdcd4 demonstrated **inhibition** of transcription from the MAP4K1 promoter. Ectopic expression of Pdcd4 in metastatic RKO cells suppressed invasion. MAP4K1 activity is functionally significant in invasion, as overexpression of a dominant negative MAP4K1 (dnMAP4K1) mutant in RKO cells inhibited not only **c-Jun activation** but also invasion. Overexpression of a MAP4K1 cDNA in Pdcd4-transfected cells rescued the kinase activity of JNK. Thus, Pdcd4 suppresses tumor progression in human colon carcinoma cells by the novel mechanism of down-regulating MAP4K1 transcription, with consequent **inhibition** of **c-Jun activation** and AP-1-dependent transcription.
ACCESSION NUMBER: 2006065252 IN-PROCESS
DOCUMENT NUMBER: PubMed ID: 16449643
TITLE: Tumorigenesis suppressor pdcd4 down-regulates mitogen-activated protein kinase kinase kinase kinase 1

expression to suppress colon carcinoma cell invasion.
AUTHOR: Yang Hsin-Sheng; Matthews Connie P; Clair Timothy; Wang Qing; Baker Alyson R; Li Chou-Chi H; Tan Tse-Hua; Colburn Nancy H
CORPORATE SOURCE: Laboratory of Cancer Prevention, Center for Cancer Research, National Cancer Institute, Frederick, MD 21702, USA.. hyang3@uky.edu
SOURCE: Molecular and cellular biology, (2006 Feb) Vol. 26, No. 4, pp. 1297-306.
Journal code: 8109087. ISSN: 0270-7306.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: NONMEDLINE; IN-PROCESS; NONINDEXED; Priority Journals
ENTRY DATE: Entered STN: 20060202
Last Updated on STN: 20060222

L1 ANSWER 2 OF 67 MEDLINE on STN
TI Regulation of axotomy-induced dopaminergic neuron death and c-Jun phosphorylation by targeted **inhibition** of cdc42 or mixed lineage kinase.
AB Mechanical transection of the nigrostriatal dopamine pathway at the medial forebrain bundle (MFB) results in the delayed degeneration of dopaminergic neurons in the substantia nigra pars compacta (SNpc). We have previously demonstrated that **c-Jun activation** is an obligate component of neuronal death in this model. Here we identified the small GTPase, cdc42, and mixed lineage kinases (MLKs) as upstream factors regulating neuronal loss and activation of c-Jun following MFB axotomy. Adenovirus-mediated expression of a dominant-negative form of cdc42 in nigral neurons blocked MFB axotomy-induced activation (phosphorylation) of MAP kinase kinase 4 (MKK4) and c-Jun, resulting in attenuation of SNpc neuronal death. Pharmacological **inhibition** of MLKs, MKK4-activating kinases, significantly reduced the phosphorylation of c-Jun and abrogated dopaminergic neuronal degeneration following MFB axotomy. Taken together, these findings suggest that death of nigral dopaminergic neurons following axotomy can be attenuated by targeting cell signaling events upstream of c-Jun N-terminal mitogen-activated protein kinase/c-Jun.
ACCESSION NUMBER: 2005683427 MEDLINE
DOCUMENT NUMBER: PubMed ID: 16336220
TITLE: Regulation of axotomy-induced dopaminergic neuron death and c-Jun phosphorylation by targeted **inhibition** of cdc42 or mixed lineage kinase.
AUTHOR: Crocker Stephen J; Hayley Shawn P; Smith Patrice D; Mount Matthew P; Lamba Wiplove R; Callaghan Steven M; Slack Ruth S; Park David S
CORPORATE SOURCE: Neuroscience Research Institute, University of Ottawa and Ottawa Health Research Institute, Ottawa, Ontario, Canada.
SOURCE: Journal of neurochemistry, (2006 Jan) Vol. 96, No. 2, pp. 489-99. Electronic Publication: 2005-11-29.
Journal code: 2985190R. ISSN: 0022-3042.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200602
ENTRY DATE: Entered STN: 20051223
Last Updated on STN: 20060218
Entered Medline: 20060217

L1 ANSWER 3 OF 67 MEDLINE on STN
TI The Janus role of c-Jun: cell death versus survival and regeneration of neonatal sympathetic and sensory neurons.

AB We investigated the functional outcome of **c-Jun activation** in sympathetic and sensory neurons of neonatal rat superior cervical ganglion (SCG) and dorsal root ganglion (DRG), respectively. Distinctly different roles of **c-Jun activation** have been suggested for these two types of neurons. In dissociated sympathetic neurons, c-Jun has been demonstrated to promote apoptosis, whereas in sensory neurons it stimulates axonal outgrowth. In organ-cultured ganglia, we found that c-Jun was activated within 24 h of explantation in both types of neurons, and that the JNK inhibitor SP600125 could mitigate this response. In both types of neurons, **c-Jun activation** was also reduced by NGF treatment. **Inhibition** of **c-Jun activation** did not affect the viability of sympathetic neurons, whereas the number of apoptotic sensory neurons increased. Furthermore, **inhibition** of c-Jun reduced axonal outgrowth from both SCG and DRG. Thus, in organ culture, **c-Jun activation** may be required for axonal outgrowth and, at least in sensory neurons, it promotes survival. The role of ATF3, a neuronal marker of injury and a c-Jun dimerization partner, was also examined. We found an ATF3 induction in both SCG and DRG neurons, a response, which was reduced by JNK **inhibition**. The reduction of ATF3 upon JNK **inhibition** was much larger in DRG than in SCG, a result which might account for the higher number of apoptotic neurons in JNK inhibitor exposed DRG. Taken together, and contrary to our expectations, neonatal sympathetic and sensory neurons seem to respond to axonal injury similarly with respect to **c-Jun activation**, and in no case was this activation pro-apoptotic.

ACCESSION NUMBER: 2005538359 MEDLINE
DOCUMENT NUMBER: PubMed ID: 16126201
TITLE: The Janus role of c-Jun: cell death versus survival and regeneration of neonatal sympathetic and sensory neurons.
AUTHOR: Lindwall Charlotta; Kanje Martin
CORPORATE SOURCE: Lund University, Department of Cell and Organism Biology, Animal Physiology Building, Helgonavagen 3B, SE-223 62 Lund, Sweden.. charlotta.lindwall@cob.lu.se
SOURCE: Experimental neurology, (2005 Nov) Vol. 196, No. 1, pp. 184-94. Electronic Publication: 2005-08-29. Journal code: 0370712. ISSN: 0014-4886.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200511
ENTRY DATE: Entered STN: 20051012
Last Updated on STN: 20051215
Entered Medline: 20051122

L1 ANSWER 4 OF 67 MEDLINE on STN
TI The role of p-c-Jun in survival and outgrowth of developing sensory neurons.
AB **c-Jun activation** has been implicated not only in neuronal apoptosis, but also in survival and regeneration. This Janus facet of **c-Jun activation** could be related to neuronal cell type or to the developmental stage of the neuron. We investigated **c-Jun activation** in E18 sensory neurons. Cultures of rat dorsal root ganglia neurons were maintained with or without the addition of nerve growth factor or the c-Jun N-terminal kinase inhibitor, (D)-JNKI1. Few dorsal root ganglia neurons survived nerve growth factor deprivation, whereas neurons supplied with nerve growth factor survived and exhibited extensive axonal outgrowth. Activated c-Jun was present in the nuclei of neurons with regenerating axons, but not in apoptotic neurons. c-Jun N-terminal kinase **inhibition** reduced the number of p-c-Jun immunoreactive and

regenerating neurons, and increased cell death. Thus, activation of c-Jun seems to be required for survival and regeneration of developing sensory neurons.

ACCESSION NUMBER: 2005516257 MEDLINE
DOCUMENT NUMBER: PubMed ID: 16189472
TITLE: The role of p-c-Jun in survival and outgrowth of developing sensory neurons.
AUTHOR: Lindwall Charlotta; Kanje Martin
CORPORATE SOURCE: Department of Cell and Organism Biology, Lund University, Sweden.. charlotta.lindwall@cob.lu.se
SOURCE: Neuroreport, (2005 Oct 17) Vol. 16, No. 15, pp. 1655-9. Journal code: 9100935. ISSN: 0959-4965.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200512
ENTRY DATE: Entered STN: 20050929
Last Updated on STN: 20051215
Entered Medline: 20051201

L1 ANSWER 5 OF 67 MEDLINE on STN
TI **Inhibition** of Rac GTPase triggers a c-Jun- and Bim-dependent mitochondrial apoptotic cascade in cerebellar granule neurons.
AB Rho GTPases are key transducers of integrin/extracellular matrix and growth factor signaling. Although integrin-mediated adhesion and trophic support suppress neuronal apoptosis, the role of Rho GTPases in neuronal survival is unclear. Here, we have identified Rac as a critical pro-survival GTPase in cerebellar granule neurons (CGNs) and elucidated a death pathway triggered by its inactivation. GTP-loading of Rac1 was maintained in CGNs by integrin-mediated (RGD-dependent) cell attachment and trophic support. Clostridium difficile toxin B (ToxB), a specific Rho family inhibitor, induced a selective caspase-mediated degradation of Rac1 without affecting RhoA or Cdc42 protein levels. Both ToxB and dominant-negative N17Rac1 elicited CGN apoptosis, characterized by cytochrome c release and activation of caspase-9 and -3, whereas dominant-negative N19RhoA or N17Cdc42 did not cause significant cell death. ToxB stimulated mitochondrial translocation and conformational activation of Bax, **c-Jun activation**, and induction of the BH3-only protein Bim. Similarly, **c-Jun activation** and Bim induction were observed with N17Rac1. A c-jun N-terminal protein kinase (JNK)/p38 inhibitor, SB203580, and a JNK-specific inhibitor, SP600125, significantly decreased ToxB-induced Bim expression and blunted each subsequent step of the apoptotic cascade. These results indicate that Rac acts downstream of integrins and growth factors to promote neuronal survival by repressing c-Jun/Bim-mediated mitochondrial apoptosis.

ACCESSION NUMBER: 2005427726 MEDLINE
DOCUMENT NUMBER: PubMed ID: 16092944
TITLE: **Inhibition** of Rac GTPase triggers a c-Jun- and Bim-dependent mitochondrial apoptotic cascade in cerebellar granule neurons.
AUTHOR: Le Shoshona S; Loucks F Alexandra; Udo Hiroshi; Richardson-Burns Sarah; Phelps Reid A; Bouchard Ron J; Barth Holger; Aktories Klaus; Tyler Kenneth L; Kandel Eric R; Heidenreich Kim A; Linseman Daniel A
CORPORATE SOURCE: Research Service, Veterans Affairs Medical Center, Denver, Colorado 80220, USA.
SOURCE: Journal of neurochemistry, (2005 Aug) Vol. 94, No. 4, pp. 1025-39.
Journal code: 2985190R. ISSN: 0022-3042.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200509
ENTRY DATE: Entered STN: 20050815
Last Updated on STN: 20050928
Entered Medline: 20050927

L1 ANSWER 6 OF 67 MEDLINE on STN
TI The neuroprotection of insulin on ischemic brain injury in rat hippocampus through negative regulation of JNK signaling pathway by PI3K/Akt activation.
AB Current studies demonstrated that cell survival is determined by a balance among signaling cascades, including those that recruit the Akt and JNK pathways. In our present work, the relationship between Akt1 and JNK1/2 was evaluated after cerebral ischemia-reperfusion in the hippocampus in a four-vessel occlusion model of Sprague-Dawley rats. This paper was based on our present and previous studies. Firstly, Akt1 had one active peak during reperfusion following 15 min ischemia. Secondly, two peaks of JNK1/2 activation occurred during reperfusion, respectively. Thirdly, the phosphorylation of JNK substrates c-Jun and Bcl-2, and the activation of a key protease of caspase-3 were detected. They only had one active peak, respectively, during reperfusion. To clarify the mechanism of Akt1 activation and further define whether JNK1/2 activation could be regulated by Akt1 through PI3K pathway, LY294002 and insulin were, respectively, administrated to the rats prior to ischemia. Our research indicated that LY294002, a PI3K inhibitor, significantly suppressed Akt1 activation. Furthermore, LY294002 significantly strengthened both peaks of JNK1/2 activation, **c-Jun activation**, Bcl-2 phosphorylation, and the activation of caspase-3 during reperfusion. In contrast, insulin, a PI3K agonist, not only obviously activated Akt1 during early and later reperfusion, but also inhibited phosphorylation of JNK1/2, c-Jun, and Bcl-2 and attenuated the activation of caspase-3. In addition, pretreatment of insulin significantly increased the number of the surviving CA1 pyramidal cells at 5 days of reperfusion. Consequently, our results indicated that the cross-talk between Akt1 and JNK1/2 could be mediated by insulin receptor through PI3K in rat hippocampus during reperfusion. This signaling pathway might play a neuroprotective role against ischemic insults via **inhibition** of the JNK pathway, involving the death effector of caspase-3.
ACCESSION NUMBER: 2005422194 MEDLINE
DOCUMENT NUMBER: PubMed ID: 16018989
TITLE: The neuroprotection of insulin on ischemic brain injury in rat hippocampus through negative regulation of JNK signaling pathway by PI3K/Akt activation.
AUTHOR: Hui Liang; Pei Dong-Sheng; Zhang Quan-Guang; Guan Qiu-Hua; Zhang Guang-Yi
CORPORATE SOURCE: Research Center for Biochemistry and Molecular Biology, Xuzhou Medical College, 84 West Huai-hai Road, Xuzhou 221002, Jiangsu, PR China.
SOURCE: Brain research, (2005 Aug 2) Vol. 1052, No. 1, pp. 1-9. Journal code: 0045503. ISSN: 0006-8993.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200510
ENTRY DATE: Entered STN: 20050810
Last Updated on STN: 20051028
Entered Medline: 20051027

L1 ANSWER 7 OF 67 MEDLINE on STN
TI Activation of the JNK-c-Jun pathway during the early phase of neuronal apoptosis induced by PrP106-126 and prion infection.

AB Prion diseases are neurodegenerative pathologies characterized by apoptotic neuronal death. Although the late execution phase of neuronal apoptosis is beginning to be characterized, the sequence of events occurring during the early decision phase is not yet well known. In murine cortical neurons in primary culture, apoptosis was first induced by exposure to a synthetic peptide homologous to residues 106-126 of the human prion protein (PrP), PrP106-126. Exposure to its aggregated form induced a massive neuronal death within 24 h. Apoptosis was characterized by nuclear fragmentation, neuritic retraction and fragmentation and activation of caspase-3. During the early decision phase, reactive oxygen species were detected after 3 h. Using immunocytochemistry, we showed a peak of phosphorylated c-Jun-N-terminal kinase (JNK) translocation into the nucleus after 8 h, along with the activation of the nuclear c-Jun transcription factor. Both pharmacological **inhibition** of JNK by SP600125 and overexpression of a dominant negative form of c-Jun significantly reduced neuronal death, while the MAPK p38 inhibitor SB203580 had no effect. Apoptosis was also studied after exposure of tg338 cortical neurons in primary culture to sheep scrapie agent. In this model, prion-induced neuronal apoptosis gradually increased with time and induced a 40% cell death after 2 weeks exposure. Immunocytochemical analysis showed early **c-Jun activation** after 7 days. In summary, the JNK-c-Jun pathway plays an important role in neuronal apoptosis induced by PrP106-126. This pathway is also activated during scrapie infection and may be involved in prion-induced neuronal death. Pharmacological blockade of early pathways opens new therapeutic prospects for scrapie PrP-based pathologies.

ACCESSION NUMBER: 2005306993 MEDLINE
DOCUMENT NUMBER: PubMed ID: 15932590
TITLE: Activation of the JNK-c-Jun pathway during the early phase of neuronal apoptosis induced by PrP106-126 and prion infection.
AUTHOR: Carimalo J; Cronier S; Petit G; Peyrin J-M; Boukhtouche F; Arbez N; Lemaigre-Dubreuil Y; Brugg B; Miquel M-C
CORPORATE SOURCE: Laboratoire 'Differenciation et Mort Neuronales', CNRS UMR 7102, case 12, Universite Paris 6, 9 quai St-Bernard, 75005 Paris, France.
SOURCE: The European journal of neuroscience, (2005 May) Vol. 21, No. 9, pp. 2311-9.
Journal code: 8918110. ISSN: 0953-816X.
PUB. COUNTRY: France
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200507
ENTRY DATE: Entered STN: 20050616
Last Updated on STN: 20050715
Entered Medline: 20050714

L1 ANSWER 8 OF 67 MEDLINE on STN
TI **Inhibition** of microglial inflammation by the MLK inhibitor CEP-1347.
AB CEP-1347 is a potent inhibitor of the mixed lineage kinases (MLKs), a distinct family of mitogen-activated protein kinase kinase kinases (MAPKKK). It blocks the activation of the c-Jun/JNK apoptotic pathway in neurons exposed to various stressors and attenuates neurodegeneration in animal models of Parkinson's disease (PD). Microglial activation may involve kinase pathways controlled by MLKs and might contribute to the pathology of neurodegenerative diseases. Therefore, the possibility that CEP-1347 modulates the microglial inflammatory response [tumour necrosis factor-alpha (TNF-alpha), interleukin-6 (IL-6), and monocyte chemotactic protein-1 (MCP-1)] was explored. Indeed, the MLK inhibitor CEP-1347 reduced cytokine production in primary cultures of human and murine microglia, and in monocyte/macrophage-derived cell lines, stimulated with

various endotoxins or the plaque forming peptide Abeta1-40. Moreover, CEP-1347 inhibited brain TNF production induced by intracerebroventricular injection of lipopolysaccharide in mice. As expected from a MLK inhibitor, CEP-1347 acted upstream of p38 and **c-Jun activation** in microglia by dampening the activity of both pathways. These data imply MLKs as important, yet unrecognized, modulators of microglial inflammation, and demonstrate a novel anti-inflammatory potential of CEP-1347.

ACCESSION NUMBER: 2005119386 MEDLINE
DOCUMENT NUMBER: PubMed ID: 15748162
TITLE: **Inhibition** of microglial inflammation by the MLK inhibitor CEP-1347.
AUTHOR: Lund Soren; Porzgen Peter; Mortensen Anne Louise; Hasseldam Henrik; Bozyczko-Coyne Donna; Morath Siegfried; Hartung Thomas; Bianchi Marina; Ghezzi Pietro; Bsibsi Malika; Dijkstra Sipke; Leist Marcel
CORPORATE SOURCE: Disease Biology, H. Lundbeck A/S, Ottiliavej 9, 2500 Valby, Denmark.. sorl@lundbeck.com
SOURCE: Journal of neurochemistry, (2005 Mar) Vol. 92, No. 6, pp. 1439-51.
Journal code: 2985190R. ISSN: 0022-3042.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200504
ENTRY DATE: Entered STN: 20050308
Last Updated on STN: 20050423
Entered Medline: 20050422

L1 ANSWER 9 OF 67 MEDLINE on STN
TI Activity deprivation-dependent induction of the proapoptotic BH3-only protein Bim is independent of JNK/**c-Jun activation** during apoptosis in cerebellar granule neurons.
AB Bcl-2-interacting mediator of cell death (Bim), a proapoptotic BH3-only protein, plays a critical role in neuronal apoptosis. Cerebellar granule neurons (CGNs) depend on activity for their survival and undergo apoptosis when deprived of depolarizing concentration of KCl. While it has been proposed that the activation of c-Jun NH2-terminal protein kinase (JNK)/c-Jun pathway contributes to the upregulation of bim gene in neurons subjected to survival signaling withdrawal, here we show that neither **inhibition** of JNK activity nor expression of dominant-negative c-Jun suppresses the expression of bim gene induced by activity deprivation in CGNs. We conclude that induction of bim gene is independent of the activation of JNK/c-Jun signaling pathway by activity deprivation during apoptosis of CGNs.

ACCESSION NUMBER: 2005038313 MEDLINE
DOCUMENT NUMBER: PubMed ID: 15664113
TITLE: Activity deprivation-dependent induction of the proapoptotic BH3-only protein Bim is independent of JNK/**c-Jun activation** during apoptosis in cerebellar granule neurons.
AUTHOR: Shi Leyu; Gong Shoufang; Yuan Zhongmin; Ma Chi; Liu Yanling; Wang Chuanfu; Li Wenming; Pi Rongbiao; Huang Shoujian; Chen Ruzhu; Han Yifan; Mao Zixu; Li Mingtao
CORPORATE SOURCE: Department of Pharmacology, Zhongshan Medical College, SUN Yat-sen University, No. 74, Zhongshan Road 2, Guangzhou 510080, China.
CONTRACT NUMBER: HD39446 (NICHD)
SOURCE: Neuroscience letters, (2005 Feb 25) Vol. 375, No. 1, pp. 7-12. Electronic Publication: 2004-11-24.
Journal code: 7600130. ISSN: 0304-3940.
PUB. COUNTRY: Ireland

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200504
ENTRY DATE: Entered STN: 20050125
Last Updated on STN: 20050419
Entered Medline: 20050418

L1 ANSWER 10 OF 67 MEDLINE on STN
TI JNK regulates the release of proapoptotic mitochondrial factors in reovirus-infected cells.
AB Reovirus-induced apoptosis is associated with activation of the proapoptotic mitogen-activated protein kinase c-Jun N-terminal kinase (JNK) and the JNK-associated transcription factor c-Jun. Here we show that reovirus-induced apoptosis and activation of caspase 3 are inhibited in cells deficient in MEK kinase 1, an upstream activator of JNK in reovirus-infected cells. **Inhibition** of JNK activity following reovirus infection delays the release of proapoptotic mitochondrial factors and the subsequent onset of apoptosis. In contrast, reovirus-induced apoptosis is not blocked by infection with adenovirus expressing dominant-negative c-Jun, and **c-Jun activation** does not correlate with apoptosis in reovirus-infected cells. This is the first report demonstrating that JNK is associated with regulation of mitochondrial pathways of apoptosis following viral infection.
ACCESSION NUMBER: 2004570091 MEDLINE
DOCUMENT NUMBER: PubMed ID: 15542665
TITLE: JNK regulates the release of proapoptotic mitochondrial factors in reovirus-infected cells.
AUTHOR: Clarke Penny; Meintzer Suzanne M; Wang Yibing; Moffitt Lisa A; Richardson-Burns Sarah M; Johnson Gary L; Tyler Kenneth L
CORPORATE SOURCE: Dept. of Neurology (B 182), University of Colorado Health Sciences Center, 4200 East 9th Ave., Denver, CO 80262, USA.. penny.clarke@uchsc.edu
CONTRACT NUMBER: 1R01AG14071 (NIA)
SOURCE: Journal of virology, (2004 Dec) Vol. 78, No. 23, pp. 13132-8.
Journal code: 0113724. ISSN: 0022-538X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200412
ENTRY DATE: Entered STN: 20041116
Last Updated on STN: 20041220
Entered Medline: 20041206